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Mechanisms of Resistance to Acetyl-Coenzyme A Carboxylase Inhibitors: a Review*

Malcolm D. Devine

Department of Crop Science and Plant Ecology, University of Saskatchewan, 51 Campus Drive, Saskatoon, Saskatchewan, Canada S7N 5A8

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Abstract: Resistance to acetyl-coenzyme A carboxylase (ACCase) inhibitors has developed in at least 10 grass weed species in recent years. In most instances, resistance is conferred by an ACCase alteration in the resistant biotypes that reduces sensitivity to aryloxyphenoxypropionate (AOPP) and cyclohexanedione (CHD) herbicides. Analysis of ACCase from many of these resistant weed biotypes suggests the presence of different mutations, each conferring a different pattern and level of resistance to various AOPP and CHD herbicides. In all cases analyzed to date, resistance is controlled by a single dominant or semi-dominant nuclear gene. In several weed biotypes, resistance is conferred by enhanced herbicide detoxification, primarily through elevated expression or activity of cytochrome P450 monooxygenase(s). This mechanism can confer cross-resistance to herbicides from other chemical classes with different modes of action. Finally, multiple herbicide resistance, i.e. the acquisition of several different resistance mechanisms, has been reported in some weed biotypes.

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1 INTRODUCTION

Two classes of herbicides, the aryloxyphenoxypropionic acids (AOPP) and cyclohexanediones (CHD) inhibit the plastidic enzyme acetyl-coenzyme A carboxylase (ACCase; E.C. 6.4.1.2).[1–3] ACCase is a key enzyme in fatty acid biosynthesis, catalyzing the condensation of acetyl-CoA with bicarbonate to form malonate in the first committed step in this pathway. ACCase inhibitors are used selectively in dicotyledonous (dicot) crops to control many grass weeds. This selectivity is based on the high sensitivity of grass ACCase to these herbicides, in contrast to the low sensitivity of dicot ACCase.[4,5] In addition, these herbicides can be used in certain cereal crops; in this case, selectivity is based on enhanced herbicide detoxification in the tolerant grass crops.[6] Finally, several grass species (*Festuca rubra* L.; *F. ovina* L. and *F. amethystina* L.) are tolerant of AOPP and CHD herbicides based on the presence of an insensitive form of ACCase.[7,8] The resistant form of ACCase in these grasses has not been characterized to date. In this paper, the mechanisms of resistance to ACCase inhibitors in weeds is reviewed.

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2 DEVELOPMENT OF RESISTANCE TO ACCase INHIBITORS

Since their introduction almost 20 years ago, ACCase-inhibiting herbicides have been used widely to control many annual and perennial grass weeds. The repeated use of ACCase inhibitors in the same fields over many

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TABLE 1
Species in which Resistance to ACCase Inhibitors has Developed

Species	*Location(s)*
Setaria viridis[a]	Western Canada
Setaria faberi[a]	Iowa, Wisconsin, USA
Eleusine indica[a]	Malaysia
Avena fatua[a]	Canada; USA; Australia; UK
Avena sterilis[a]	Australia; UK
Alopecurus myosuroides[a]	UK; Spain; Germany; France
Lolium rigidum[a]	Australia; Spain
Lolium multiflorum[a]	Oregon, USA; UK
Digitaria sanguinalis	Wisconsin, USA
Sorghum halepense[a]	Mississippi, USA
Echinochloa colona	Costa Rica

[a] Resistance conferred by reduced ACCase sensitivity in at least one biotype.

years has led to the development of resistance to these herbicides. To date, resistance has been confirmed in at least 10 weed species in Europe, North and Central America, Eurasia and Australia (Table 1).[6,9] Although there has been little by way of systematic surveys for ACCase resistance, a detailed analysis in Manitoba, Canada in an area with a history of repeated use of ACCase inhibitors indicated that 20 out of 30 fields surveyed contained resistant biotypes of *Avena fatua* L.[10]

3 MECHANISMS OF RESISTANCE TO ACCase INHIBITORS

3.1 Resistance based on reduced ACCase sensitivity

In the majority of weed biotypes, resistance to ACCase inhibitors is conferred by reduced sensitivity of the target enzyme to these herbicides.[6,9] This is normally demonstrated by extraction and partial purification of ACCase from young leaf tissue, followed by an enzyme activity assay in the presence of a range of herbicide concentrations. Dose-response results from typical assays of ACCase from resistant and susceptible biotypes of *Setaria faberi* Herrm. and *Eleusine indica* Gaertn. are shown in Figs 1 and 2;[12,16] a more complete description of the ACCase sensitivity of various resistant weed biotypes is shown in Table 2. The results in Table 2 indicate that the two resistant *Setaria viridis* (L.) Beauv. biotypes are not equally resistant to all herbicides. In addition, it is clear that the two resistant biotypes are not the same, but differ in the level of resistance to specific herbicides. For example, biotype UM8 has a comparable level of resistance to all herbicides tested (R/S ACCase I_{50} ratios ranging from 30 to 60), whereas biotype UM131 is very resistant to sethoxydim, less resistant to fenoxaprop and diclofop,and only marginally resistant to clethodim (Table 2).[11,12] It is likely that different mutations occur in the ACCase from the two resistant biotypes, conferring different patterns of resistance to different inhibitors. A third *S. viridis* biotype (designated UM137) was identical to UM131, but was also resistant to dinitroaniline herbicides. In this case, it is likely that resistance originally developed to dinitroaniline herbicides and that subsequent selection with ACCase inhibitors led to the selection of plants with a second resistance mutation.

High-level resistance to sethoxydim has also been identified in a biotype of *S. faberi*. Resistance in this biotype can also be attributed to reduced ACCase sensitivity, similar to that observed in the *S. viridis* biotype UM131 (Fig. 1; Table 2).[12] ACCase from a sethoxydim-resistant maize (*Zea mays* L.) line, now commercially available in the USA, is also extremely resistant to sethoxydim and less resistant to other ACCase inhibitors, suggesting it may carry a similar resistance mutation.[13,14]

The ACCase sensitivity of several other resistant weed biotypes is shown in Table 2. A resistant biotype of *E. indica* developed in Malaysia following repeated use of ACCase inhibitors.[15] ACCase from this biotype was very resistant to fluazifop, less resistant to fenoxaprop and sethoxydim, and only marginally resistant to clethodim (approximately four-fold) (Table 2; Fig. 2).[16] It is unclear whether this level of resistance confers any meaningful resistance at the whole-plant level. Treat-

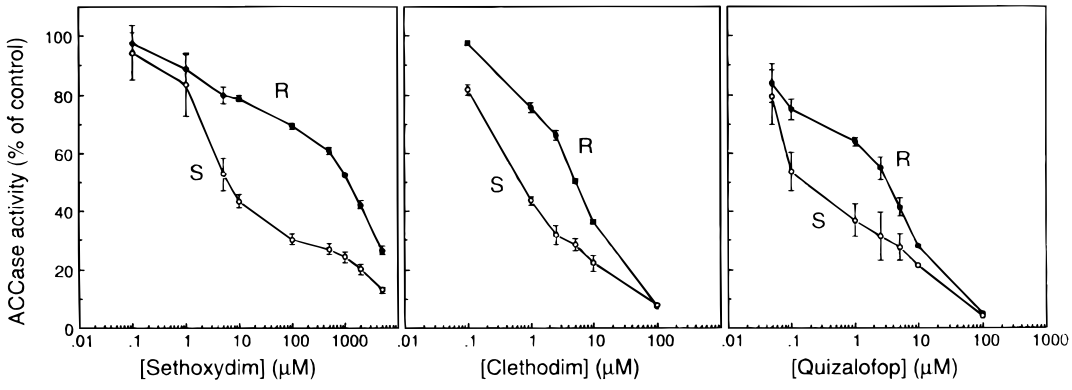

Fig. 1. Inhibition of ACCase from (S) susceptible and (R) resistant biotypes of *Setaria faberi*. From Ref. 12; used with permission.

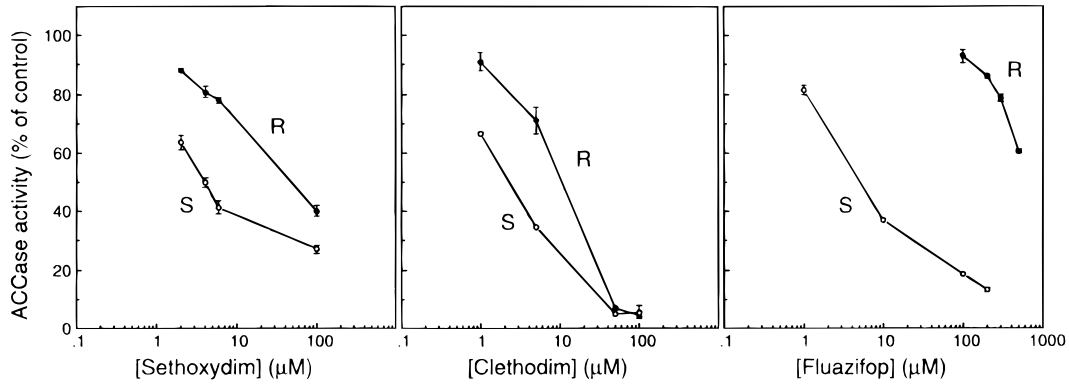

Fig. 2. Inhibition of ACCase from (S) susceptible and (R) resistant biotypes of *Eleusine indica*. From Ref. 16; used with permission.

ment with 50 g ha^{-1} clethodim under controlled environment conditions provided complete control of both the susceptible and resistant biotypes.[15] Similarly, treatment of the resistant *S. faberi* biotype with the normal application rate of clethodim provided satisfactory control of this biotype.[12] However, detailed dose-response data are not available for either resistant biotype.

Two *Avena* species, *A. fatua* L. and *A. sterilis* L. (both commonly referred to as wild oat) occur as agricultural weeds in temperate regions. Resistance to ACCase inhibitors has developed in both species in Australia, Canada, the USA and England, with reduced ACCase sensitivity to various herbicides as the predominant mechanism (Shukla, A., Moss, S. & Devine, M. D., unpublished).[17–19] As in the other species in which resistance has developed, the resistant *Avena* biotypes appear to carry different ACCase mutations, conferring different levels of resistance to AOPP and CHD herbicides (Table 2).

Initial results with an AOPP- and CHD-resistant biotype of *A. fatua* from western Canada indicated that resistance was not conferred by reduced ACCase sensitivity.[20] However, further research failed to reveal a convincing mechanism of resistance (see Section 4). Recently, detailed re-analysis of ACCase from this and other *A. fatua* biotypes has shown that resistance is due to reduced ACCase sensitivity.[19] It appears that enzyme extraction conditions play an important role in the results obtained, and that optimization of extraction conditions can reveal resistant forms of ACCase that may be masked or altered when extracted under different conditions. Moreover, the optimum conditions for ACCase extraction appear to differ between biotypes; the optimum conditions for one biotype did not reveal an insensitive ACCase in a second biotype, but further modification of the extraction conditions did reveal that the second biotype also contained a resistant form of

TABLE 2
Summary of Sensitivity of ACCase from Various Resistant Weed Biotypes to AOPP and CHD Herbicides.

		R/S I_{50} [a]					
Species	*Biotype*	*Fenoxaprop*	*Diclofop*	*Fluazifop*	*Sethoxydim*	*Tralkoxydim*	*Clethodim*
Setaria viridis	UM8	48·3	46·7		50·0	31·3	31
	UM131	6·9			423		2·4
S. faberi		3·0			192		5·7
Avena fatua	UM1	9·8	10·1		105	10·1	
	UM33	10·5	1·2		5·0	1·7	
A. sterilis	SAS 1	52		9·2	8·1	5·7	
Lolium rigidum	SLR 3		>36·5	⩾2·9	7·8	>9·5	
	SLR 31		6	55	26	14	
	WLR 96		>217	>6·9		6	
	VLR 69		29	4·3	1·2	1·0	
L. multiflorum			27·7		0·9		1·5
Eleusine indica		24·5		>90	20·4		4·4
Sorghum halepense	Leland		6·7	15	4·7		1·7
Alopecurus myosuroides	Lincs	>17	5·6	>58	3·3	>4·5	

[a] ACCase I_{50} for each herbicide in the resistant biotype/ACCase I_{50} in the susceptible biotype. See text for references.

ACCase.[19] These results suggest that caution should be exercised in interpreting the results of ACCase assays, especially when the results indicate no difference in sensitivity between resistant and sensitive biotypes.

Intensive herbicide use has led to the widespread selection of herbicide-resistant biotypes of *Lolium rigidum* Gaud. and *L. multiflorum* Lam., many of which are resistant to ACCase inhibitors. Several of these have been characterized, showing reduced ACCase sensitivity to be an important resistance mechanism (Table 2).[21–25] Again, the evidence points to a family of ACCase mutations, each conferring a different resistant phenotype.

Finally, target site-based resistance has been identified in biotypes of *Alopecurus myosuroides* Huds. from Europe (L. M. Hall, pers. comm.) and *Sorghum halepense* Pers. from the USA (Table 2) (Marles, M. A. S. and Devine M. D., unpublished results, and Reference 26).

Plants contain multiple forms of ACCase that differ in their sensitivity to herbicides. For example, maize contains two ACCases (designated ACCase I and ACCase II) which are similar in molecular mass (227 and 219 kD, respectively) but differ in their affinity for certain substrates.[27] ACCase I is sensitive to haloxyfop and sethoxydim, whereas ACCase II is relatively insensitive. Similarly, susceptible *L. multiflorum* contains two ACCases, one of which is very sensitive to diclofop and the other insensitive (I_{50} values 0·2 and >125 μM, respectively).[28] A resistant biotype of *L. multiflorum* also contained two ACCases, but ACCase I in the R biotype was less sensitive to inhibition by diclofop ($I_{50} =$ 7·0 μM). Similar results have been obtained in a resistant *A. sativa* biotype (Shukla, A. and Devine M. D., unpublished results). These results suggest that resistance to ACCase inhibitors in these biotypes is conferred by reduced herbicide sensitivity of ACCase I, the predominant plastidic form of the enzyme. Inheritance studies have shown that ACCase-based resistance to AOPP and CHD herbicides is governed by a single semi-dominant nuclear gene, and that resistance in weed biotypes that differ in their pattern of resistance to different herbicides is encoded at the same gene locus.[24,29,30]

The mutations conferring target-site based resistance to ACCase inhibitors have not been identified. However, it is likely that research currently under way in several laboratories will reveal the identity of a family of point mutations, each conferring a specific resistant phenotype. The results are likely to be analogous with acetolactate synthase mutations, where many point mutations have been identified, each associated with a unique pattern of resistance to different ALS inhibitors.[9] In the case of ACCase, it appears that resistant mutants may be grouped as follows: (a) high-level resistance to sethoxydim, low to other herbicides (e.g. *Setaria viridis* UM131, *S. faberi* and *A. fatua* UM1 in Table 2); (b) high-level resistance to fluazifop, low to others (e.g. *L. rigidum* SLR 31, *E. indica* and *A. myosuroides* biotype Lincs in Table 2); (c) relatively high-level resistance to AOPP herbicides, very low or none to CHD herbicides (e.g. *A. fatua* UM33, *L. rigidum* VLR 69 and *L. multiflorum* in Table 2) and (d) one or more additional mutations, conferring intermediate levels of resistance to various herbicides. Comparison of ACCase sequences within and between these groups of resistant biotypes and from susceptible biotypes will lead to identification of the mutations conferring the different resistance patterns.

3.2 Resistance based on enhanced herbicide metabolism

Enhanced herbicide metabolism is a mechanism of resistance to ACCase inhibitors in several weed biotypes, particularly in *L. rigidum* and *A. myosuroides*.[23,31,32] Resistance in these biotypes is often conferred by elevated expression or activity of cytochrome P450 monooxygenase(s), which is commonly involved in the initial step of herbicide metabolism in plants. Herbicide resistance in these biotypes can be synergized by piperonyl butoxide or 1-aminobenzotriazole, inhibitors of plant Cytochrome (Cyt) P450 monooxygenases.[23] Depending on the form(s) of Cyt P450 involved, this mechanism can also confer resistance to other classes of herbicide with different modes of action. For example, elevated Cyt P450 activity confers resistance to sulfonylurea, phenyl urea, *s*-triazine and CHD herbicides in a single *L. rigidum* biotype.[23]

Recently, several additional weed biotypes have been identified that are resistant to one or more ACCase inhibitors and cross-resistant to other herbicides with different modes of action (e.g. biotypes of *A. fatua*, *Echinochloa colona* (L.) Link.). Although no evidence has yet been obtained, it is likely that resistance in these biotypes is conferred by enhanced metabolism to the different classes of herbicides.

3.3 Other resistance mechanisms

Earlier research on resistance to ACCase inhibitors in one *L. rigidum* and one *A. fatua* biotype suggested that resistance was due not to reduced ACCase sensitivity or enhanced herbicide metabolism, but was correlated with apparent differences in herbicide effects on plasma membrane function.[20,33] Treatment with diclofop rapidly depolarized the plasma membrane electrogenic potential (Em) in resistant and susceptible biotypes of both species, but only the resistant biotypes were able to regenerate the Em when diclofop was removed from the bathing solution. Further experiments with the resistant *A. fatua* biotype showed that diclofop and tralkoxydim uptake into isolated plasma membrane

vesicles and protoplasts was 15–30% lower than in the susceptible biotype;[34] in contrast, uptake was equal into isolated chloroplasts of the resistant and susceptible biotypes (Wang, X. and Devine, M. D., unpublished results). These results suggested a resistance mechanism associated with the plasma membrane.

However, despite considerable effort, no experimental evidence has been obtained clearly identifying such a mechanism. It is unlikely that the small reduction in herbicide uptake could confer the level of herbicide resistance observed (6- and 13-fold to diclofop and tralkoxydim, respectively)[35] in the resistant *A. fatua* biotype. In addition, no differences were found in the protein or lipid content of the plasma membranes.[36] Diclofop does affect the trans-membrane proton gradient in both susceptible and resistant tissues,[20,33,37] but the relevance of this to its phytotoxicity *in vivo* is unclear. The effect on the plasma membrane Em is intriguing, and may be involved in the antagonism between diclofop and auxinic herbicides such as 2,4-D,[38] but its importance as a resistance mechanism is questionable. In addition, our recent finding that the *A. fatua* biotype in question has a resistant form of ACCase which was not detected in the original assays[19] suggests that resistance in some of the biotypes with the hypothesized 'membrane-based' resistance mechanism may also be conferred by reduced ACCase sensitivity.

4 CONCLUSIONS

Resistance to ACCase inhibitors is an increasing problem in many species in many parts of the world. It is clear that this resistance can arise relatively easily, following six to 10 years of selection pressure. Current indications are that fitness is not reduced by the resistance mutations and that the resistant weeds are not at a selective disadvantage when growing in the absence of herbicide treatment.[39,40] It might be expected, therefore, that the continued use of these herbicides as the predominant grass weed control measure will give rise to many more resistant weed populations. In most cases resistance is conferred by target-site mutations that confine the resistance to ACCase inhibitors; other grass herbicides (e.g., dinitroanilines, acetanilides, etc.) continue to provide satisfactory control of these biotypes. However, multiple target-site mutations have developed in some biotypes, severely limiting chemical weed control options.

Where resistance is due to enhanced herbicide metabolism, cross-resistance to other chemical classes of herbicides is of concern. In these cases resistance may be conferred to classes of herbicides that have never been used on the particular weed biotype. Again, this may seriously limit chemical weed control options. The judicious use of ACCase inhibitors in conjunction with other classes of herbicide and non-chemical control methods will be important in maintaining the long-term usefulness of these herbicides. Future research must be aimed at developing weed control strategies (herbicide rates, frequency of use, use of mixtures, etc.) that maximize the useful life of these herbicides for the long-term benefit of crop protection.

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